Internal Parasites of Poultry

BY EVERETT E. WEHR
AND JOHN F. CHRISTENSEN [1]

A THOROUGH ACCOUNT of the principal parasites of poultry in the United States, including practical measures for preventing some of the heavy annual losses from this source.

JAMES E. RICE, formerly head of the Poultry Husbandry Department of Cornell University, who has been called the father of poultry husbandry in the United States, once made the statement that there was no way to save the poultry industry except through a scientific approach to the control of poultry diseases. The significance of this statement becomes apparent with the realization that losses from poultry diseases in the United States have been estimated by Government authorities to be approximately 100 million dollars annually.

That the presence of disease has been responsible for the curtailment of poultry raising in many areas cannot be questioned. A few years ago poultrymen in many sections of the United States were forced to abandon the raising of turkeys because of the prevalence of blackhead, caused by a protozoan parasite. Only a drastic change in poultry-husbandry practices made turkey production again a profitable enterprise in these areas.

Farmers are losing 18.8 percent of their poultry because of disease, or 1 out of every 5 birds, according to C. M. Ferguson of the University of Ohio. This is a tremendous loss, since statistics indicate that chickens are kept on 85 percent of the farms in the United States at the present time.

Internal parasitism is usually more insidious, and therefore not so noticeable as specific diseases, many of which are deadly in their effects and spectacular in nature, but parasites constitute a real menace to successful poultry raising. Unlike bacterial diseases, the majority

[1] Everett E. Wehr is Zoologist and John F. Christensen is Associate Protozoologist, Zoological Division, Bureau of Animal Industry.

of the parasitic diseases do not result in early fatalities. The protozoan diseases, blackhead and coccidiosis, are exceptions, since the destruction of the intestinal mucous membrane in the former and of the liver tissue in the latter are attended by a high rate of mortality.

Poultry raisers are demanding more and better control measures for both parasites and diseases. Though only a few satisfactory drugs for the removal of poultry parasites have been found, practical and reliable control measures are not lacking. Sanitation, when properly carried out, has proved to be one of the most effective and practical means of reducing parasitism in poultry flocks.

Since measures for the control of internal parasites in poultry are largely preventive and apply to almost the whole group of parasites, they will be discussed first in this article. The little that can be done in the way of curative treatment will also be included in the first part of the article. Descriptions of the large numbers of poultry parasites and the injuries they cause will follow the section on control.

CONTROL OF POULTRY PARASITES

Many species of parasites must spend a part of their developmental cycle outside the body of the host or they cannot continue to exist. In the case of poultry parasites, with few exceptions, the stage away from the bird is spent in the feces. Some of the protozoan parasites, however, spend this developmental stage in the blood stream, and these parasites are therefore not eliminated through any of the body openings of the host. Their escape from the body depends on their being removed by bloodsucking insects.

In their infective stages, parasites are introduced into susceptible hosts in a number of ways: (1) By means of contaminated litter and soil. (2) By means of contaminated food and water. (3) By means of carriers; birds that have had a light infection or have survived a more severe one may carry the organisms within their intestinal tracts for long periods of time and spread the parasites in their discharges. (4) By intermediate hosts; insects such as flies, mosquitoes, and beetles, or other low forms of animal life such as snails and slugs, in which a part of the development of the parasite takes place, are eaten by susceptible hosts, and as a result infection is set up. (5) By mechanical means; animals and human beings may carry infective material on their feet and thus spread the parasites from one pen to another—attendants have been known to carry infective material from an infected pen to a sanitary one; contaminated chicken coops and other equipment may be a source of infection when carelessly moved from place to place or used for healthy birds.

Prevention Versus Cure

The old adage that an ounce of prevention is worth a pound of cure is just as applicable to poultry parasites as it is to human diseases. After a disease has once gained a foothold in a flock, far more time and money are usually spent in getting rid of the disease than would

have been necessary to keep the premises free of it. In addition, the mortality may be high before the disease is finally checked.

Preventive measures include sanitation, disinfection, hygiene, and management.

Sanitation and Hygiene

Sanitation means establishing an environment in which the possibility of infection with disease-producing organisms is reduced to a minimum.

The first essential is the proper selection of the poultry site. The soil should be of a sandy or gravelly nature in order to provide for good drainage. To take advantage of natural drainage, the site should preferably be located on a gentle slope. If the lay of the land or the nature of the soil renders natural drainage impossible, artificial drainage must be resorted to. Since moisture is essential to the development of most parasites in their free-living stages, the presence of surface water, which the birds are apt to drink, must be regarded as unhealthful. Pools of water in the poultry yards should be immediately filled in or drained.

Poultry houses or shelters are essential for the protection of the birds against inclement weather, including rain, storms, and extremes of temperature. The excreta, or body wastes, of the birds which collect in the houses or shelters must be properly handled, or they may serve as a source of infection with parasites and other diseases. Irrespective of the type of poultry house built, it is of the utmost importance that it possess certain features of design and arrangement to facilitate cleaning and to keep the birds well and strong. The floors and walls should be constructed of material that is impervious to moisture and easy to clean and will exclude vermin of all types. Floors of dirt or wood are seriously objectionable because they are difficult to clean and disinfect; concrete floors are much more desirable. The roosts and nests should be simply constructed so that they may be taken apart easily for cleaning and disinfecting. (See Farmers' Bulletin 1070 (*10*).)[2] The house should be so constructed as to insure the entrance of an abundance of direct sunlight, which is important to the health of the fowls as well as destructive to bacteria and certain parasites.

The hygienic condition of poultry yards is of tremendous importance to the health of the birds. It is not so convenient to collect and dispose of body wastes in the poultry runs as in the houses. For this reason, the small overcrowded poultry runs too frequently observed are apt to receive a larger amount of wastes than the ground can adequately take care of. When a disease is once introduced in such a place, it quickly becomes epizootic, that is, like an epidemic among human beings, and soon all the birds have contracted it. This condition exists on many farms in the United States, and because of the prevalence of disease under such conditions, the individual farm flock rarely pays for itself.

[2] Italic numbers in parentheses refer to Literature Cited, p. 1037.

Permanent Quarters or Rotation

Keeping birds on the same ground year after year will ultimately cause the soil to become a more or less continual hotbed of infection if parasitized or diseased birds are present in the flock. The eggs of parasites are known to live in the soil from year to year, and contaminated soil serves as an important source of infection for the intermediate hosts of poultry parasites.

What is the remedy for such a situation? The course that a poultryman may choose depends very largely on the size of the flock, the available space for rearing the birds, the amount of money that can be allotted for poultry equipment, and the type of soil. If only a small area of land is available, it may be best to raise the birds in confinement. This system has the advantage that the birds may occupy the same quarters year after year. In order to provide adequate sunlight, a small wire-covered sun porch may be constructed in front of the house, or there may be a small fenced-in yard covered with cinders or other porous material.

Where a considerable area of land is available for poultry raising, the practice of rotating the fowls has been followed with reasonably good success. This system consists of using enclosed poultry yards which are occupied by the birds intermittently in order to prevent excessive contamination. The four-yard system is the one most widely advocated and is probably the one best suited for general farm practice. A plot of land—the acreage depends on the number of birds to be raised, figuring about 650 to 800 birds per acre—is fenced and cross fenced so as to have four equal-sized pens. The shelter or house is located in the center of the plot and built with a door opening directly into each pen. The birds are placed in lot No. 1, kept there for a month or two, and then moved into lot No. 2. The practice of moving the birds at regular intervals from one pen to another is continued until all four pens have been occupied, when the birds are again placed in pen No. 1. Immediately following the removal of the birds from one of the pens, the ground should be prepared and planted to some green crop or left idle to undergo self-sterilization. In the latter case the contaminated soil is left undisturbed so that the sun, wind, and cold can act directly on the parasites present in it. Some poultrymen plant the lots to a permanent crop, such as alfalfa. Such a crop furnishes abundant green feed for the growing birds, and this makes it less likely that they will pick up contaminated material from the soil. The house and adjacent grounds should be cleaned at least once a week, and oftener if necessary.

Manure Disposal

Every poultry owner is faced with the problem of disposing of the body wastes from his birds in a sanitary manner. To allow the excreta to accumulate in large quantities either in the house or in the yard is to bring into operation the law of nature that no species of animal can exist for very long in intimate contact with its own body wastes without endangering its health through the contraction of

disease. Wild birds and animals are not restricted in their range as are the species raised in more or less confinement, hence wild creatures are less apt to pick up infections from their excretions.

Under present conditions in the poultry industry it is often necessary to raise several thousand birds together, and proper steps must be taken to remove the body wastes at frequent intervals and to dispose of them in a sanitary manner if mortality from disease is to be kept at a low level. The body wastes from farm flocks can readily be disposed of by having them hauled to the fields and spread thinly over the land for use as fertilizer. Poultry manure, when properly handled, is an excellent fertilizer for garden and field crops. The body wastes from flocks raised in back yards or in communities where poultry raising is intensively practiced cannot be so easily disposed of as on the farm and must be taken care of in some other way. In some heavily populated areas, poultry raisers have been known to store the manure from their flocks in one or more centrally located storage sheds and sell it. Poultry manure usually retails for a very good price.

To properly preserve the fertilizing value of poultry manure, it must be stored in a suitable screened-in shed with a cement floor and a roof to keep out rain and snow. Manure stored in this manner is also protected from flying and crawling insects and other forms of animal life that may serve as carriers or intermediate hosts of poultry parasites. Just what effect the storage of poultry manure in piles has on the livability of the eggs of poultry parasites and of coccidial oöcysts has not been definitely determined.

Different Types of Poultry Should Be Raised Separately

It has been demonstrated repeatedly that in order to prevent the spread of parasitic and other poultry diseases the different types of poultry should be raised separately and in small flocks. Observations have shown that turkeys serve as carriers of gapeworms and transmit gapeworm disease to little chicks, while older chickens are almost entirely resistant to gapeworm infection under range conditions. On the other hand, Tyzzer (*46*) shows that chickens carry the organisms of blackhead in their intestinal tracts, and young turkeys may contract the disease by exposure to infected chickens or to areas infected by them and usually die in large numbers from the disease. Only in exceptional cases do the chickens show symptoms of the disease, and infected chickens usually recover and remain carriers of the organisms for an indefinite period.

It is also dangerous for chickens to associate with pigeons. Levine (*34*) was successful in producing severe experimental infections in chickens with the pigeon capillarid, *Capillaria columbae*. Wehr (*52a*) has reported that chickens heavily infected with *C. columbae* showed symptoms of emaciation, diarrhea, and listlessness and that such an infection usually resulted in death.

77434°—51——65

What To Do In Case of An Outbreak

In case of an outbreak of a poultry disease, an early diagnosis is essential in order that the proper control measures may be employed. Unfortunately, in many places the services of a poultry-disease diagnostician or a competent veterinarian are not available. In such cases, the State agricultural experiment station should be consulted immediately as to the advisability of shipping birds to its poultry department or veterinary science department for a diagnosis. Should it be necessary to ship sick birds to a distant diagnostic laboratory, it may be several days before a reply is received. In the meantime, losses may continue, and the disease, if acute in character, may gain such a foothold that control measures, when finally applied, will be of little use. Since it is urgent that the disease be checked as soon as possible after its appearance, it is suggested that the following first-aid measures be put into effect immediately in an attempt to control it before it reaches epizootic proportions.

The first requirement in bringing an outbreak of disease under control is to remove all visibly sick birds from the flock, confine them in a room or house separate from the healthy birds, and burn the carcasses of any birds that have died. The healthy birds should if possible be moved to a clean house and clean grounds, but if this is impossible, the body wastes that have accumulated in the house should be disposed of in a sanitary manner, and the house and all its equipment should be thoroughly cleaned and disinfected with some suitable disinfectant, such as hot water or hot lye solution (see the article on Disinfection and Disinfectants, p. 179). As soon as the house and equipment have been cleaned and disinfected, the healthy birds should be put back in the house and confined there until the symptoms have subsided in the sick birds. During this period of confinement, the healthy birds should be watched carefully, and any of them that become sick should be removed immediately and placed with the sick birds. The houses occupied by both the healthy and sick birds should be thoroughly cleaned daily, preferably dry-cleaned, and the litter burned. In case of diarrhea, a mild laxative should be given to all the birds. For flock treatment, 1 pound of Epsom salts dissolved in about 2 gallons of water will make enough medicated liquid for 100 birds. The birds should be kept warm and be disturbed as little as possible, and crowding should be avoided. The feed should be placed in sanitary hoppers and the watering devices protected with wire frames, so that the birds will have less chance to contaminate the drinking water. If the same person must tend both the sick and the healthy birds, the sick birds should be attended to last in order to prevent the mechanical transfer of the causative agent of the disease to the healthy birds. The attendant should be provided with a pair of rubbers for use in each pen and a pan of disinfectant (a weak lysol solution) for dipping the soles of the shoes or rubbers before and after entering the pen or house. Visitors who have been handling poultry elsewhere should not enter the poultry house or yard.

Medicinal Agents and Their Use

While anthelmintic (worm-expelling) treatment has proved to be applicable to many kinds of livestock and has served to reduce parasitism in them to the point where it apparently does little or no harm, the use of medicinal agents in the control of poultry parasites has not met with any great success. Despite the large amount of experimental work done on the control of poultry parasites by means of medication, effective and practical drugs for the control of such economically important diseases as coccidiosis, blackhead, and tapeworms are still lacking. Although some progress has been made in the treatment of other parasitic diseases of poultry, satisfactory drugs for the control of many of the roundworms are also lacking.

Figure 1.—Equipment for and method of administering barium antimonyl tartrate to chickens infected with gapeworms.

To be satisfactory, a drug designed for the removal of parasites from poultry must be inexpensive, relatively easy to administer, highly effective, and relatively nontoxic. The value of the individual bird is ordinarily so low that the cost of administering drugs to fowls, unless mass treatment can be resorted to, is in most cases prohibitive.

Effective drugs have been discovered for the removal of the gapeworm, the large roundworm, and the cecum worm, and the treatment will be discussed briefly.

Hall and Shillinger (*17*), and others, have reported that carbon tetrachloride is an effective drug for the removal of the large intestinal roundworm, *Ascaridia galli*. Ackert and Graham (*3*) found carbon tetrachloride highly efficacious in removing the large intestinal roundworm from young chickens with apparently no ill effects.

For the control of the cecum worm, Hall and Shillinger (*17*) recommend rectal injections of a mixture of oil of chenopodium and olive or cottonseed oil. McCulloch and Nicholson (*37*) stated that

phenothiazine, given in either single or repeated doses, was very effective for the removal of cecum worms from chickens. Roberts (*40*) reported that phenothiazine was effective for the removal of the cecum worm but not of the large intestinal roundworm.

Wehr, Harwood, and Schaffer (*53*) found that barium antimonyl tartrate given as an inhalant successfully removed a large percentage of the gapeworms (*Syngamus trachea*) from chicks, turkey poults, and grown turkeys. For treatment, the infected birds are confined in a tight container into which the powder is introduced through an opening near the top by means of a dust gun (fig. 1). The worms in the trachea are killed by the dust inhaled by the birds.

It has been demonstrated that trichomoniasis (infection with trichomonads) of the lower digestive tract of poultry can be successfully treated by means of heat therapy. Olsen and Allen (*38*) successfully treated a number of turkeys infected with *Trichomonas gallinarum* by placing them in a thermostatically controlled cabinet for periods ranging from 1 to 2 hours. The internal body temperature of the birds was raised from 2° to 6° above the normal temperature of 106.5° F. by maintaining an air temperature of approximately 104° F., and a relative humidity of 60 to 70 percent within the treatment box. These investigators found that three treatments at intervals of every other day were sufficient to check the disease, but in advanced cases it was sometimes necessary to administer as many as six treatments. As soon as the body temperature returned to normal, the birds were removed from the cabinet and placed in wire-bottomed cages. Forced feeding of liquid mash was resorted to when the birds refused to eat voluntarily.

Occasionally a bird failed to respond to the heat treatment. Those that did respond usually began to eat voluntarily, gained in weight, and behaved like normal, active birds after the second or third treatment. Several adult turkey hens were treated and later laid several clutches of eggs.

A few of the treated birds were killed at different stages of recovery following treatment, and post mortem examinations disclosed that many of the liver lesions (tissue injuries) had almost completely disappeared and others were in the process of healing. Cultures made from partly healed lesions showed no trichomonads. A relatively large percentage of the untreated birds died from trichomoniasis.

No satisfactory treatment for trichomoniasis of the upper digestive tract has been developed.

THE PROTOZOAN PARASITES OF POULTRY

The protozoan parasites of poultry that are significant as disease producers and therefore of concern to poultrymen belong to two major groups, the flagellates and the Sporozoa. The flagellates include actively moving protozoa equipped with from one to many whiplike hairs, or flagella, which are used to propel the organism through the fluids in which they live. The Sporozoa, or spore-forming organisms, are almost exclusively parasitic and are charac-

terized by the absence of definite organs for movement and by their peculiar, often complex life histories.

A species of the flagellated organisms scientifically designated as *Hexamita meleagridis* is suspected of playing an important role in the production of a severe intestinal infection in young turkeys known as infectious catarrhal enteritis (inflammation of the intestines). The part played by another flagellate, *Histomonas meleagridis*, in the production of so-called "blackhead" disease, or enterohepatitis (literally, inflammation of the intestines and the liver), of turkeys is well known. At least two species of flagellates of the genus *Trichomonas* are associated with intestinal disturbances of young turkeys, one producing caseating necrotic lesions (injuries characterized by the presence of cheesy matter and dead tissue) in the crop and esophagus, or gullet, and the other responsible for large cecal (blindgut) and liver lesions similar in character to those of blackhead; these conditions have been designated as trichomoniasis of the upper digestive tract and of the lower digestive tract, respectively.

The most important sporozoan parasites of poultry are the coccidia, which are of such tremendous economic importance as the cause of coccidiosis in chickens that they are discussed in a separate article in this Yearbook (p. 1041). Two other sporozoan parasites that are arousing considerable attention and are believed to be responsible for malarialike diseases of young ducks and turkeys are identified by most investigators as species of *Leucocytozoon*.

With the exception of the coccidia, the protozoan parasites of poultry have their greatest significance, from the disease standpoint, in turkeys. Other domestic birds such as chickens, ducks, geese, guinea fowl, and pigeons frequently harbor similar or identical parasites, a few of which appear to be pathogenic, or disease-producing, to their hosts, while most are tolerated with little or no inconvenience. In the following discussion of specific diseases known or believed to be caused by protozoan parasites, primary emphasis is placed on turkeys, the host birds in which these disorders reach their largest proportions. The general principles of protozoan parasitism and parasite control, however, apply equally to other poultry.

Infectious Catarrhal Enteritis of Turkeys

Infectious catarrhal enteritis (hexamitiasis) of turkeys, associated with *Hexamita meleagridis*,[3] is becoming increasingly important in the United States. This disease has been known to exist in California for some time. It is apparently increasing in occurrence and severity with the expansion in turkey production, the increased crowding of birds on ranches probably affording greater opportunities for turkeys to acquire the infection. The technical name "infectious catarrhal enteritis" indicates that the disease is a contagious in-

[3] Among other designations, the disease has been described recently as a "trichomoniasis," but Hinshaw, McNeil, and Kofoid (*21*) presented convincing field and experimental evidence which definitely eliminated *Trichomonas* as the causative agent and implicated another protozoan parasite, a species of *Hexamita*. Hinshaw and McNeil (*20*) gave the specific name *meleagridis* to the *Hexamita* which they stated was "the causative agent of infectious catarrhal enteritis" in turkeys. In view of this recent information, there seems to be justification for designating the infection as hexamitiasis. The present discussion of the disease is based on the work of these investigators.

testinal inflammation characterized by abnormally heavy secretion from the mucous glands of the affected intestine.

Hexamitiasis is primarily an acute infection of the upper part of the small intestine of turkey poults between the ages of 1 and 12 weeks, with the greatest death loss occurring at 3 to 5 weeks of age. The symptoms of an acute outbreak are not very specific, being in general similar to those of other acute intestinal diseases. Sick poults are listless and droopy, walk with a stilted gait, and often have a watery or foamy diarrhea. The birds may continue to eat but fail to digest and assimilate food normally, with rapid loss of flesh as a consequence. In individual birds, the acute infection runs a short course of 1 to 6 days after symptoms appear, but in large flocks the peak of mortality during an outbreak occurs in 7 to 10 days after the first sick birds are observed. Most birds that survive acute infections fail to recover from the emaciation and remain stunted. Mortality from acute outbreaks on California ranches has been reported to vary from 20 to 90 percent. Subacute infections sometimes occur, characterized by listlessness and loss of weight in affected birds. The majority of the birds that survive acute infections, as well as those with subacute infections, become carriers and are potential sources of infection to young birds.

The chief sign of the disease seen on post mortem examination of birds that have died from acute hexamitiasis is catarrhal inflammation of the upper part of the small intestine. The intestinal contents are thin and abnormally watery. The intestinal walls have lost tone and may be thin and flabby, with local distended areas having an inflamed mucous membrane. *Hexamita meleagridis* may be consistently demonstrated under the microscope in scrapings from the affected intestinal wall and occurs in particularly large numbers in the inflamed distended areas. In severe cases the flagellates may occur throughout the entire small intestine. The enormous numbers of *Hexamita meleagridis* found in poults with enteritis have not been observed in healthy birds.

Hexamita meleagridis is a microscopic, spindle-shaped, flagellated protozoan measuring on the average about one twenty-five hundredth of an inch in length. It is readily distinguished in structure and movements from the trichomonads, which are often found associated with it in the cecal discharges of the same bird, by the absence of an undulating membrane along the edge of the body, by the absence of an axostyle, or "tail," at the hind end, and by the fact that the *Hexamita* organism moves rapidly in a fairly straight line rather than jerkily. This parasite reproduces by simple longitudinal splitting, each organism dividing frequently to form two individuals. In both diseased and carrier birds living flagellates are continually discharged in the droppings. Susceptible poults may acquire the infection by swallowing feed or soil contaminated with droppings containing the parasites.

Both field and experimental evidence point to *Hexamita meleagridis* as the sole or principal cause of catarrhal enteritis of turkey poults.

It has been definitely shown by investigators in California that

the adult turkey is the primary source of infection. The causative agent of the disease has been transmitted to quail and from quail to turkeys. Field evidence indicates that quail may serve as an important carrier of the infection in some regions.

Histomoniasis, or So-Called "Blackhead" Infection

Histomoniasis, or enterohepatitis, is an acute, highly fatal disease of turkeys attributed to infection with the protozoan parasite *Histomonas meleagridis*. Though the acute infection in chickens is usually mild and transitory, chickens have an important part in the complete picture of histomoniasis because they are established as carriers following primary infection and thus serve as a source of infection for susceptible turkeys. Since the principal sites of infection in diseased turkeys are the walls of the ceca and the liver, the disease is technically termed "infectious enterohepatitis," which indicates that it is a contagious infection involving the intestine and liver. Under farm conditions, histomoniasis probably includes most of the so-called blackhead of turkeys. Dark discoloration of the head is not a constant symptom of the disease, however, and may be produced by disorders of the circulatory system due to other causes. It is therefore probably more appropriate to designate the disease as histomoniasis. The unfortunate nonspecific term blackhead might well be eliminated from the veterinary vocabulary. Although the technical name may seem difficult to laymen, usage would soon give it the same currency as such names as "coccidiosis" and "trichomoniasis."

Histomoniasis has forced the abandonment of turkey raising in many parts of this country. Application of the principles unearthed in recent researches, however, particularly those of Tyzzer and his associates, has again made turkey production a successful enterprise in these areas. Tyzzer's report (*45*) on histomoniasis summarized the detailed information on the modes of infection of susceptible birds and the form, structure, and life history of the parasite that is the basis of the present conception of the disease.

Turkeys are susceptible to histomoniasis at any age up to maturity. The disease develops within 2 to 3 weeks after the susceptible birds acquire infective organisms by ingesting feed or soil contaminated with droppings from infected birds. The course of the disease is rapid, death usually occurring soon after the development of symptoms. Sick birds are weak, listless, and droopy, and usually have a sulfur-colored diarrhea. Dark discoloration of the head, as already noted, is not a constant symptom. Death may sometimes occur without visible symptoms. Histomoniasis in older birds is less acute than in young poults, the illness usually being more prolonged and the mortality lower. The great majority of the younger birds that develop the disease die, the mortality often being 100 percent.

On post mortem examination, birds dying from histomoniasis show greatly enlarged ceca, or blind pouches, that contain cheesy masses of tissue debris often infiltrated with blood. The cecal walls are thickened and congested and show large, ulcerlike lesions. The

liver also usually appears somewhat enlarged and is blotched with characteristic, slightly sunken, reddish-gray lesions of various sizes. Thin sections from the cecal walls, when mounted on slides and stained appropriately for examination under the microscope, show *Histomonas* organisms among the surface cells lining the walls. The living parasites may often be recovered from the characteristic liver lesions and demonstrated under the microscope.

The histomonads are peculiar flagellated protozoa with certain amoeboid, or amoebalike, tendencies. As they occur in the cecal contents of carrier birds, they are normally more or less rounded bodies that usually show amoeboid movements of the protoplasm (that is, they are capable of pushing out temporary arms of protoplasm from their surfaces) as well as rhythmic rotatory movements produced by the beating of a single locomotor whip, or flagellum. They measure on an average about one two-thousandth of an inch in length, although individuals may be considerably larger and possess as many as four flagella.

Immediately upon establishment in the ceca of a susceptible host bird, the histomonads multiply rapidly, invade the mucous membrane of the cecal walls, and become rounded tissue forms. Invasion of tissues is usually brief in the chicken, which recovers with only slight inconvenience, though deaths from histomoniasis among chickens have been known, and typical cecal and liver lesions have been seen at autopsy. The histomonads usually persist in the cecal contents indefinitely, and the chicken is established as a carrier of the infection. In the more susceptible turkey, invasion of tissues is so energetic that the histomonads gain access to the blood stream and are carried to the liver, where the characteristic lesions are produced. The infected birds usually die from severe cecal and liver infection (enterohepatitis). The few turkeys able to survive the acute infection also become carriers. Both chickens and turkeys with acute or carrier infections are sources of infection to new susceptible hosts, discharging living histomonads regularly in their droppings. When shed from infected birds, the histomonads are either free in the feces or housed in some manner not yet clearly understood within the eggs of the cecal worm, *Heterakis gallinae*, which are also eliminated in the feces after being produced by the adult worms in the ceca of the host birds. New hosts acquire the infection by swallowing feed or soil contaminated with droppings containing the parasites, either free or within the eggs of the cecal worm.

Leucocytozoan Disease of Ducks and Turkeys

Turkey poults under 12 weeks of age and ducklings 10 days to a few weeks old are most susceptible to leucocytozoan disease. The general symptoms are common to both kinds of host birds, and therefore the infections in the two need not be differentiated. In the young birds the disease strikes suddenly, with acute symptoms lasting only 2 or 3 days. Sick birds first lose appetite, become droopy, and have a tendency to sit down from weakness or exhaustion. They seem to be thirsty and drink large amounts of water. Their breath-

ing becomes heavy, and they may crawl instead of walking. In the later stages of severe infections, the birds are excitable and when disturbed may fall over, lapse into a coma during which the breathing is labored, and die. Some very sick birds die after only a few minor convulsions. Blood removed from the vein of a bird sick with this condition shows greatly enlarged spindle-shaped cells, somewhat larger than the normal red blood cells, which contain the parasitic organisms. The most characteristic lesion at autopsy is an enlarged and blackened spleen. The birds that recover from acute infections may either remain permanently stunted or show no serious effects, but most become carriers of the infection. Mortality from the acute disease is reported to range from 0 to 100 percent in ducklings and from 10 to 50 percent in turkeys.

It was originally believed that the parasites invaded the white cells, or leucocytes, of the blood of infected birds, and for this reason they were named *Leucocytozoon*. Some later investigators believed, however, that the red corpuscles rather than the leucocytes were the host cells. This question is still not settled definitely. Whatever these host cells may prove to be, they are greatly altered by parasitism with the leucocytozoa. The parasitized host cells become elongated to 4 or 5 times their normal length, with ends tapered to points. The full-grown parasites, usually one to each host cell, are elongated oval or bean-shaped structures that almost completely fill the parasitized cells exclusive of the tapered ends. The ultimate result of this parasitism is the destruction of the host cells, with anemia as a consequence. It has been suggested that the cause of the respiratory difficulties observed in birds with advanced, severe leucocytozoan disease may be due to mechanical blocking of the small capillaries of the lungs with these large parasites.

The large parasites inside the cells of the circulating blood of the host birds represent only a stage of the life history of *Leucocytozoon*. They develop further only when ingested by certain species of *Simulium*, or common blackflies, that feed on the blood of the infected birds. In the engorged blackflies, the leucocytozoa pass through a definite cycle of development, and within a few days small infective forms known as sporozoites are present in the salivary glands. Some of these sporozoites are expelled into the blood stream of susceptible young birds on which the blackflies feed. In ducks, the sporozoites are said to penetrate the surface cells lining the small capillaries of the lungs, liver, and spleen and to undergo generations of multiplication there that result in the production of enormous numbers of parasites. Eventually these products of multiplication are liberated into the blood stream and invade cells of the circulating blood, where they may be detected by means of stained blood smears. The entire life history has been determined to require only 2 to 3 days in the blackflies and 9 or 10 days in the host birds.[4]

Trichomoniasis of the Upper Digestive Tract of Turkeys

Trichomoniasis (that is, infection with trichomonads) of the upper digestive tract is characterized by the presence of peculiar cheesy

[4] Further details of research are given in citations *23*, *24*, *39*, *42*, and *48*.

lesions of dead tissue piled up to as high as approximately two-tenths of an inch above the surface of the mucous membranes of the esophagus and crop. The entire upper digestive tract is studded with small grayish-white nodules, or lumps, resembling the pustules found in nutritional roup. The mucous membrane is entirely destroyed, and an examination of the cheesy lesions or retained fluid of the upper digestive tract discloses countless numbers of flagellates. The lesions usually end abruptly at the zone dividing the esophagus from the proventriculus, or true stomach, indicating perhaps that the gastric juice of the true stomach may be detrimental to the survival of these organisms.

Sick birds are characterized by a depressed appearance, loss of appetite, sagging wings, emaciation, and drooling at the mouth. The droppings are usually of the consistency of water and contain large numbers of the flagellated parasites. Young birds may die as soon as 1 day after the appearance of symptoms, but older birds may linger for several weeks. In older birds the region surrounding the crop usually appears depressed and sometimes pendulous. Although certain other diseases may produce similar symptoms, birds that make repeated attempts to swallow, extend the head and neck, and retain the crop fluid should be suspected of trichomoniasis. Often diagnosis can be confirmed by inspection of the mouths of sick birds, since the lesions frequently occupy visible portions of the upper digestive tract. Ordinarily mortality is slight, but it has been reported to range as high as 73 percent in mature flocks to 87 percent in young flocks.

Individuals of *Trichomonas gallinae* are roughly egg-shaped flagellates measuring on an average one twenty-five hundredth of an inch in length. They are extremely active trichomonads capable of rapid forward spiral locomotion produced by the beating of the flagella at the front end and movements of the protoplasmic flange, or undulating membrane, extending along the edge of the body. It is generally believed that these upper-digestive-tract trichomonads are distinct from those inhabiting the ceca and lower intestine of turkeys. They differ in certain aspects of structure and behavior, in their respective sites of localization within the digestive tracts of the host birds, and apparently in their disease-producing capabilities. As with the other protozoan infections of turkeys, a certain percentage of the adult birds become carriers of the trichomonads and thus serve as sources of infection to susceptible birds. Presumably infection of new hosts occurs through ingestion of contaminated feed, soil, or drinking water with living trichomonads discharged from infected birds either in the droppings or in the discharges from the mouth.[5]

Trichomoniasis of the Lower Digestive Tract of Turkeys

Turkeys are victims of another disease, which was believed by Allen (*7*) to be due to *Trichomonas gallinarum*, a flagellate commonly found in the ceca and lower intestine of chickens, turkeys, guinea fowl, and probably other domestic fowls. Infection with these para-

[5] See citations *14, 19, 29, 32, 33, 43, 44,* and *49* for further details of research.

sites is common in healthy chickens and turkeys, but disease symptoms develop with much greater frequency and severity in the latter. According to Allen (*8*) the infection resembles histomoniasis in producing lesions in the ceca and liver of turkeys, but these lesions are characteristically different from those of histomoniasis. The disease may be designated tentatively as "trichomoniasis of the lower digestive tract" to distinguish it from *T. gallinae* infection of the esophagus and crop.

Birds experimentally infected with *Trichomonas gallinarum* sometimes develop the acute type of infection, characterized by diarrhea and occasional mortality in the young turkeys a few days after inoculation, but more often they develop a chronic type of the disease, the symptoms of which do not appear for several weeks after infection. Under certain conditions not yet fully understood, these trichomonads may produce an enterohepatitis, with lesions distinct from those of histomoniasis. Liver lesions have not been observed in experimentally or naturally infected birds under 3 months of age. The turkeys that develop the disease gradually and progressively lose their thrifty condition, become droopy, often have intermittent attacks of diarrhea characterized by pale-yellow droppings, and usually die. The slow, chronic development of the disease is sharply contrasted with the acuteness of histomoniasis. The importance of this type of trichomoniasis has not been fully determined. The insidious, chronic nature of the infection, its tendency to produce isolated losses rather than mass mortality, and the possible confusion of the specific lesions with those of histomoniasis have probably permitted the disease to go undetected. The studies of Allen indicate that it may prove to be of considerable importance.

The cecal and liver lesions are the most obvious changes to be observed at post mortem examination of birds dead of the disease. The liver lesions are the most clearly defined, being irregular, granular, often slightly elevated areas of cheesy appearance, quite distinct from the rounded, nongranular, noticeably depressed lesions of histomoniasis. Similar lesions are present on the cecal walls, and often one or both ceca contain cores of cheesy, dead material infiltrated with blood. In naturally infected turkeys, the lesions may occur separately or together with those of histomoniasis, and there appears to be basis for the belief that in the past the lesions of the two distinct diseases have often been collectively diagnosed as blackhead lesions. In the interest of accurate diagnosis and proper evaluation of their relative importance the two infections should be carefully differentiated.

Allen (*7*) expressed the opinion that the common cecal trichomonads of turkeys are identical with *Trichomonas gallinarum*, described previously from the chicken. These trichomonads are on an average considerably smaller but much more active than the histomonads, which may often occur in the same cecal contents from infected birds. They vary from nearly spherical to distinctly pear-shaped, and their average length is approximately one thirty-five hundredth of an inch. They are typical trichomonads, each individual possessing several whips, or flagella, and an undulating membrane along the edge

of the body. The combined movements of these flagella and the undulating membrane give a jerky type of locomotion with little forward progression, characteristically different from the rapid forward movements of *T. gallinae*.

As known at present, the life history of *Trichomonas gallinarum* is apparently simple. The trichomonads in the cecal contents of the host birds feed by absorption from the fluids in the lumen, or cavity, of the ceca, and each individual periodically reproduces by asexual splitting to form two trichomonads. Infected birds regularly shed living trichomonads in the cecal droppings. No resistant, or cyst, forms have been observed in the course of the life history; and if no cysts are formed, it is evident that young birds must pick up living trichomonads very soon after they are discharged from infected birds, since these flagellates are extremely susceptible to drying and changes in temperature. Once ingested by susceptible birds in feed, soil, or drinking water, the parasites must survive the passage through the alimentary tract in order to become established in the lower intestine and ceca of the new host birds.

METAZOAN PARASITES OF POULTRY

TREMATODES

Flukes, or trematodes, are small, flattened, unsegmented worms which as adults are parasites of both invertebrates and vertebrates. Most flukes are internal parasites, usually inhabiting the intestinal tract, lung, liver, or some other internal organ. A few, however, have been classified as external parasites, since they are found in the skin and similar locations.

At least three species of flukes have been reported as parasitizing poultry in the United States, but none of them is of any great economic importance. One species, *Collyriclum faba*, is an external parasite that occurs in the skin of domestic and wild birds. The other two species, *Prosthogonimus macrorchis* and *Psilostomum ondatrae*, are internal parasites, the former occurring in the bursa Fabricii (a glandular sac), and the egg-forming organs and the latter in the proventriculus.

The cystic or skin fluke, *Collyriclum faba*, has been reported from the domestic fowl in Minnesota, and it has also been found in wild birds in Massachusetts, Maryland, Minnesota, Wisconsin, New Jersey, New York, and Michigan.

These flukes produce small, hard, cystlike structures, usually in the region of the vent. However, they have also been found in the skin just in front of the anus, in the chest region, over the lower surface of the abdomen and breast, around the beak on both the external and the internal surfaces, on the neck, and on the crop.

The smooth, shiny, grayish-white cysts are one-twelfth to two-fifths inch (2 to 10 mm.) in diameter and contain two approximately hemispherical flukes, in contact along their flattened surfaces. A dark-brown or almost black substance exudes from the opened cyst, which contains the minute eggs of the parasite.

The life history of the fluke is not completely known. It is be-

lieved that some snail is its first intermediate host, and evidence points very definitely to nymphs of dragonflies as the second intermediate host. The eggs escape from the cysts through an opening in the cyst wall and are scattered wherever the bird goes. Masses of degenerating and decaying cysts have been noted on chickens which, when they are removed or have dropped off, apparently serve to disseminate the disease, since the dead areas contain myriads of eggs. When brought into contact with water, the eggs hatch and apparently continue their development in a snail. The prevalence of this parasite in sparrows, crows, nuthatches, and other land birds, however, suggests that some land invertebrate may serve as the first intermediate host. Because of its prevalence in the English sparrow, it is thought that this bird may serve as an important disseminator of the disease.

The effects of the parasite on the fowl, aside from the possible slight injury to the general health of the bird and a disfigurement of the skin, which lowers the market value, are not noteworthy.

Proventriculitis (inflammation of the true stomach) in chickens may be due to a small fluke, *Psilostomum ondatrae*, commonly found in muskrats and water birds. The life history of this fluke is not known. The only report of the parasite's occurring in domestic fowl in the United States is from Colorado. Twenty-nine deaths in a flock of 42 White Leghorns 8 weeks old and 8 deaths in a flock of Plymouth Rock pullets were attributed to infection with the parasite. Infected birds develop inappetence (lack of appetite) and lethargy and gradually waste away. After several days of sickness, death results, apparently from starvation. Post mortem findings are an enlarged and ulcerated proventriculus, a deep reddening around the openings of the glands, and, in severe cases of infection, a grayish exudate, or discharge, on the surface of the glandular stomach. The flukes apparently do not burrow into the proventriculus but produce the irritation by their presence on the surface of the mucous membrane.

A small, reddish-colored fluke, nearly one-fourth of an inch long, occurs in the bursa Fabricii and oviduct of the domestic chicken and duck in the Great Lakes region. This parasite has been reported by Kotlan and Chandler (*30*) from the oviduct of a wing-pinioned duck in Michigan. Although believed by some investigators to be identical with *Prosthogonimus pellucidus*, a species of fluke occurring in a similar location in the domestic chicken and duck in Europe, this fluke has been described by Macy (*35*) as a new species, *P. macrorchis*.

Owing to its location within the reproductive organs, *Prosthogonimus macrorchis* may be responsible for serious losses, due to reduced or complete stoppage of egg production among laying hens. The symptoms shown by infected fowls are dullness, loss of weight, and a greatly decreased egg production. Controlled experiments have shown that uninfected birds laid nearly 10 times as many eggs as the infected hens. In the oviduct, the parasites are responsible for acute inflammation and the formation of abnormal eggs. The irritation resulting from the presence of these flukes in the oviduct

causes a reversal of the peristaltic movements, which results in broken yolks, albumen, bacteria, and parasitic material entering the abdominal cavity and giving rise to acute peritonitis, or inflammation of the abdominal lining. Kotlan and Chandler (*30*) described the pathological changes resulting from the presence of these worms as—

> heavy emaciation and anemia; fibrinous peritonitis, with a large amount of sticky, yellow exudate, containing large masses of egg-yolk and albumen material; a number of red-colored, live flukes were found in the exudate. The ovary showed a number of diseased, collapsed ovules, containing grayish-yellow, egg-yolk-like material mixed with fibrin and pus. Some of the ovules were apparently ruptured. The oviduct was greatly distended, its serous coverings showing a more or less pronounced reddish discoloration. The lumen of the oviduct contained a large amount of albumen material forming ovoid clots of about one to two centimeters in diameter; the mucosa was covered with a sticky exudate consisting in the main of albumen, blood and fibrin.

The life cycle of the parasite involves two intermediate hosts, snails and dragonflies. Macy (*36*) found that the snail *Amnicola limosa porata*, which is common in the lakes of Michigan, Wisconsin, Minnesota and other Northern States, served as the first intermediate host. He succeeded in infecting dragonfly nymphs by placing a number of cercariae of *Prosthogonimus macrorchis* (the form of the organism found in snails) in Syracuse watch glasses containing the young dragonflies.

Kotlan and Chandler (*31*) demonstrated experimentally that chickens can become infected with the adult flukes by feeding them dragonfly nymphs containing cysts of *Prosthogonimus* species. It is believed that ducks are the normal hosts of *P. macrorchis* and the domestic hen is the abnormal host, as the latter loses its infection in 3 to 5 weeks. The rate of growth of the adult fluke is relatively slow in ducks and chicks, but more rapid in the oviduct of the hen. The fluke develops only in the oviducts of laying hens and has been occasionally found in hen's eggs.

Cestodes

Tapeworms, or cestodes, are flattened or ribbon-shaped worms composed of numerous segments or divisions. The head, neck, and a small number of the front segments are usually much narrower than the remaining portion of the worm, which grows from the neck backwards, so that the segments farthest removed from the head are the oldest from the standpoint of development. The terminal segments of the fully developed tapeworm may be filled with eggs; these are known as gravid segments and are the ones usually found in the droppings of infected birds.

Several species of tapeworms inhabit the small intestines of fowls. Each species usually shows some preference for a certain part of the small intestine to which to attach itself and develop. If tapeworms are present in large numbers, however, specimens may be found attached to portions of the intestine other than the one normally preferred.

So far as has been ascertained by experimentation, all poultry tapeworms pass the earlier part of their development in one of the

lower animals. These so-called intermediate hosts include houseflies, snails, slugs, ants, earthworms, grasshoppers, sandhoppers, and others.

Intermediate hosts become infected with young tapeworms by swallowing the gravid, or egg-bearing, segments which have been passed in the droppings of infected birds. Within the body cavity of the intermediate host, the bladder worm, or young tapeworm, has the appearance of a sac filled with liquid in the center of which is seen the head of the adult tapeworm. The head contains four cup-shaped cavities, or suckers, and a number of hooks surround the front end.

When a susceptible bird host swallows an intermediate host containing these bladder worms, they attach themselves to the intestinal wall and begin to develop segments, which appear first just back of the head in the so-called neck or growing region.

The nodular tapeworm, *Raillietina echinobothrida*, produces nodules or tuberclelike bodies in the subserous and muscular coats of the walls of the posterior (hind) portion of the small intestines of chickens and other fowls. These lesions closely resemble those of avian tuberculosis, and it is important that a careful examination be made before a positive diagnosis is given. The diagnosis should not be difficult, since nodules in the wall of the small intestine in the absence of tapeworms may be considered as being due to the tubercle bacillus and not to the nodular tapeworm. The tapeworm may be quite small and may be overlooked in a hurried or cursory examination. In case of doubt, the affected intestine should be opened and washed carefully in a stream of water. The washed intestine is then placed in a dish of water deep enough to cover it; if tapeworms are present, they will be seen hanging to the mucous membrane. This discovery, in the absence of lesions in the liver or other organs, would warrant the diagnosis of tapeworm disease.

Jones and Horsfall (*26*, *27*) showed that the ants *Tetramorium caespitum* and *Pheidole vinelandica* naturally harbored bladder worms of this tapeworm and also those of another closely related species, *Raillietina tetragona*. When these two bladder worms were fed to young chickens, the latter became infected with the adults of the cestodes. Joyeux and Baer (*28*) reported finding bladder worms of *R. echinobothrida* in naturally infected ants, *Tetramorium semilaeve*, in the region of Marseilles, France.

The proliferating, or branching, tapeworm, *Hymenolepis cantaniana*, occurs in the small intestines of chickens, quail, pheasants, turkeys, and peafowl.

Alicata and Jones (*6*) found that the small dung beetle, *Ataenius cognatus*, served as an intermediate host of this tapeworm. As found in the infected beetle, the young tapeworm consists of numerous branches with a few terminal buds which represent completely developed bladder worms. From 2 to 3 weeks are required for the bladder worm to develop into the adult tapeworm in the avian host.

The broad-headed tapeworm, *Raillietina cesticillus*, attaches itself by preference to the anterior (front) and middle portions of the small intestine of the chicken, guinea fowl, and turkey. This tapeworm is probably one of the most common cestodes of domestic fowls

in the United States. It may be readily distinguished from other species of tapeworms infecting poultry by the broadly developed head which carries a double row of 400 to 500 delicate, hammer-shaped hooks; the suckers are weakly developed and devoid of spines.

Several species of beetles belonging to the families Scarabaeidae, Tenebrionidae, and Carabidae have been shown experimentally to serve as intermediate hosts for *Railletina cesticillus*. The meal beetles, *Tribolium castaneum* and *T. confusum*, which are commonly found infesting poultry feeds, have been shown recently to serve in this capacity.

Ackert and Reid (*4*) demonstrated experimentally that chickens 2½ to 5 months of age are more resistant to infection with this species of tapeworm than younger birds and that a reduction in the blood sugar and hemoglobin contents of the blood resulted from such infections. Harwood and Luttermoser (*18*) reported that the growth of Rhode Island Red and White Leghorn chicks was retarded by infections with the tapeworm.

The minute tapeworm *Davainea proglottina* usually inhabits the duodenal region (the first part of the small intestine) of the chicken, turkey, and occasionally other birds. In heavy infections, individual tapeworms may be found as far back as the yolk stalk. This tapeworm has been reported from widely separated areas in the United States, but most often from the Eastern States. It is one of the smallest tapeworms found infecting poultry. A fully developed specimen measures about one-fiftieth to three twenty-fifths of an inch in length and is composed of two to five segments which gradually increase in length and breadth as the worm matures; the last segment may be larger than all the rest of the parasite. Because of its small size, this tapeworm is frequently overlooked.

The life cycle of *Davainea proglottina* was first demonstrated experimentally by Grassi and Rovelli (*15*) who showed that the common garden slug, also called the gray field slug, *Agriolimax agrestis*, could be successfully infected with the bladder worms of this tapeworm. In addition to this slug, many other species of slugs and two species of snails, *Polygyra thyroides* and *Zonitoides arboreus*, have been incriminated experimentally as intermediate hosts of the tapeworm. The gray field slug is very common in those sections from which *D. proglottina* has been reported and probably plays a very important role in the perpetuation of the disease wherever it and the parasite occur together.

This species has been considered by a number of investigators to be one of the obviously dangerous tapeworms infecting poultry. Heavily infected birds are said to become lethargic and waste away. On post mortem examination, the intestinal mucous membrane appears thickened, which may be due to hemorrhages, and the intestine contains a large quantity of mucus, which tends to be fetid. Leg weakness has been attributed to infections with this tapeworm, but its true relationship to this condition is still unknown.

Domesticated fowls are hosts of a number of other species of tapeworms. *Hymenolepis carioca* is a very common cestode of poultry in the United States. It is a small tapeworm measuring 1⅕

to 3⅛ inches long, threadlike, and very fragile; the segments break off easily when handled. It sometimes occurs in large numbers in chickens and turkeys, but it has very little, if any, effect on the growth rate of young chicks. This species of tapeworm utilizes the dung beetles, *Onthophagus hecate*, *Aphodius granarius*, *Choeridium histeroides*, and others as intermediate hosts.

Metroliasthes lucida, a common cestode of turkeys, measures as much as 8 inches (20 cm.) in length. It lacks a rostellum (beak), and the suckers are devoid of spines. Jones (*25*) found that the eggs of this tapeworm would develop to the bladder worm stage in grasshoppers.

Amoebotaenia sphenoides is principally a parasite of chickens. This tapeworm measures only from one-twelfth to one-sixth inch (2 to 4 mm.) long; the head has a single row of 14 hooks and is followed by a short neck. The 18 to 20 segments that follow the neck gradually increase in size up to the fourteenth and then gradually decrease. It has been demonstrated that a species of earthworm, *Ocnerodrilus africanus*, served as an intermediate host of this tapeworm. More recently, the earthworms *Helodrilus foetidus*, *Pheretima pequana*, and *Allolobrophora chloritica* have been assigned to this role. Chickens in Texas, Kansas, and Michigan have been reported as being infected with this species of tapeworm.

Under normal conditions this parasite probably causes very little damage to fowls.

Choanotaenia infundibulum, a cestode parasite of the duodenum, or forward part of the small intestine, of chickens, turkeys, and several species of wild game birds, is frequently met with in chickens and turkeys in the United States. Houseflies, grasshoppers, and several species of beetles have been reported as intermediate hosts of this tapeworm.

Nematodes

Roundworms, or nematodes, are usually elongated, cylindrical, unsegmented worms which vary from only a small fraction of an inch to several inches in length. They likewise differ greatly in habitat, having been found in poultry in almost every organ.

On the basis of their life histories, the roundworms of poultry may be divided into two general groups: (1) Those transmitted directly from bird host to bird host and (2) those requiring an insect or some other lower animal for their complete development.

The first type of life cycle is considered the simplest, for its completion involves merely the swallowing by a host animal of the embryonated roundworm eggs (those containing embryos, which are infective) with the food and water or bits of soil. The embryo roundworm hatches in the intestinal tract, and direct development to the adult stage usually takes place there. Soon after hatching, the young of many of the species of roundworms of poultry penetrate into the mucosa, or lining of the intestinal tract, and usually spend a number of days there before reentering the intestinal cavity. In the case of the gapeworm, the young worms leave the alimentary tract and

77434°—51——66

wander through various organs of the body before settling down permanently in the trachea, or windpipe.

In addition to the roundworms that pass part of their development in insects or some other form of animal life, thus requiring a true intermediate host, a third group is recognized in which the infective stage is transmitted either directly, through the medium of contaminated food and water, or indirectly, by swallowing some insect or other animal in the body of which the infective stage has encysted. The gapeworm of poultry is an example of the latter group.

Crop Worms

Domestic fowls are susceptible to infection with at least three species of crop worms, namely, *Capillaria annulata*, *C. contorta*, and *Gongylonema ingluvicola*. The first two species are commonly known

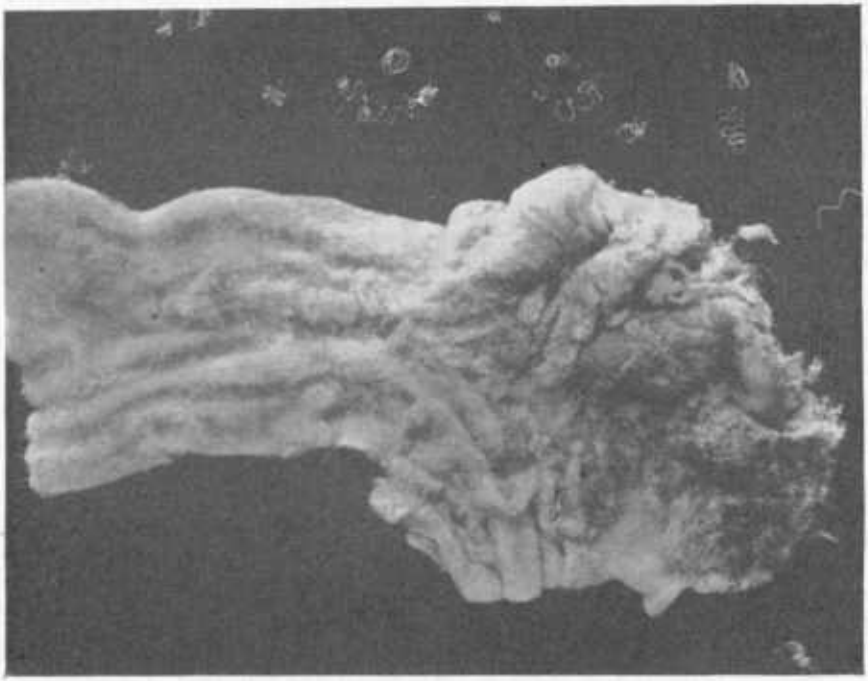

FIGURE 2.—Crop and esophagus of a bird heavily infested with the crop worm, *Capillaria annulata*

as capillarid worms, threadworms, or hairworms, while the last has been called the gullet worm.

These crop worms are long and slender, varying in length from half an inch to 2 or more inches. They bury themselves in the mucous membrane of the crop and esophagus, or gullet, in tortuous burrows, from which it is often difficult to remove them whole (fig. 2). In heavy infections, these worms may be found in the

undilated portion of the esophagus as well as the dilated portion or crop.[6]

The life histories of the two threadworms are known, but that of the gullet worm is yet to be discovered. The transmission of *Capillaria contorta* was found to be direct by Cram (*12*). At about the same time (1936) Wehr (*50*) discovered that *Capillaria annulata* required the earthworm as an intermediate host.

The chief injury produced by the crop worms is a thickening of the wall and an enlargement of the glands of the crop and esophagus. In heavy infections the crop wall is greatly thickened, highly inflamed, and congested, and the mucous membrane is loose and torn.

Stomach Worms

The proventriculus, or true stomach, is the site of infection by two parasitic roundworms, *Dispharynx spiralis* and *Tetrameres americana*.

The spiral stomach worm, *Dispharynx spiralis*, has been observed in the proventriculus of the chicken, turkey, guinea fowl, pigeon, and a few wild gallinaceous birds (the same order to which domestic poultry belong). In certain sections of the United States, particularly California and Texas, pigeons have been found to be very heavily infected with this parasitic roundworm.

The adult worms are short and thick and usually are curved or rolled in the form of a spiral; the hind end of the male is very tightly coiled. At the site of infection, the nematodes are usually found with their heads buried deeply in the wall of the proventriculus. Tumors are usually formed at the site of the attachment of the worms, and in heavy infections the wall of the glandular, or true, stomach becomes enormously and uniformly thickened as well as ulcerated.

Cram demonstrated experimentally that the pill bug serves as the intermediate host of the spiral stomach worm. Whether it is the principal intermediate host for the spread of the parasite under natural conditions is not known.

The globular roundworm, *Tetrameres americana*, is strikingly different in appearance from most roundworms. The male worm is very small and in general resembles other nematodes, but the female is globular and bright red. The two sexes likewise differ in their location within the stomach. The female worms, apparently when quite young, enter the tubular glands (Lieberkühn's glands) of the stomach wall, leaving only the hind part of the body, including the vulva, protruding into the stomach cavity. The male lives free in

[6] Wehr (*51*) observed that each of these three species of roundworms, when viewed in their normal position in the mucosa of the esophagus, displayed a different body contour. This discovery afforded a reasonably accurate method of identifying the worms at the site of the infection without resorting to a detailed microscopic examination of each worm. All three species assume a twisted position in the mucosa, but in the case of the gullet worm the perspective is one of a series of folds, approximately uniform in size and shape, following one another in close succession and usually extending in a straight course. In the two species of *Capillaria*, the body shape consists of a series of irregularly shaped folds. However, *Capillaria annulata* may be readily distinguished from *Capillaria contorta* by its much smaller size. In case of doubt the particular worm may be removed from its burrow and examined microscopically for the presence of a cuticular swelling directly back of the head which identifies it as *Capillaria annulata*.

the cavity of the stomach and apparently enters Lieberkühn's glands only for a short time to mate with the female.

Infective larvae of *Tetrameres americana* have been recovered from the body cavities of the grasshoppers *Melanoplus femur-rubrum* and *M. differentialis* 42 days after experimental infection. Chickens to which infected grasshoppers were fed later became infected with the adults of this stomach worm. Barber (*9*) stated that *T. americana* was the cause of a serious catarrhal condition in chickens in Guam. In heavy infections the walls of the proventriculus become so swollen that the cavity is almost obliterated.

Gizzard Worms

Of the parasitic roundworms occurring underneath the thick, horny lining of the gizzard of poultry, only two species are of economic importance. These worms burrow through the horny lining of the gizzard and bury themselves, sometimes deeply, in the muscles beneath. The gizzard worm of chickens and turkeys, *Cheilospirura hamulosa*, selects the anterior and posterior (front and back) portions of the gizzard, regions in which the horny covering is thin and soft, while the gizzard worm of ducks and geese, *Amidostomum anseris*, may be found generally throughout this organ.

In life these worms are reddish in color, indicating perhaps that they are bloodsuckers. The damage they cause to the gizzard may be so severe that this organ cannot function properly; thus they interfere with the digestion of the bird.

Cheilospirura hamulosa requires an intermediate host for its complete development. Several species of grasshoppers and numerous beetles, including the common meal beetle, *Tribolium castaneum*, have been found experimentally to serve in this capacity.

In lightly infected birds the lining of the gizzard may show a slight ulcerative condition, which may involve the muscular tissue as well. Soft nodules inclosing the nematodes are sometimes found in the muscular portion of the gizzard, especially in the thinner parts. In heavy infections a large part of the posterior portion of the gizzard may become enlarged and frequently loses its natural shape. The symptoms vary with the degree of infection. Mild infections are scarcely detectable, whereas severe ones are said to produce anemia and emaciation.

Amidostomum anseris frequently occurs underneath the horny lining of the gizzard of ducks and geese, sometimes in large numbers. Cram (*11*) reported a severe outbreak of this disease among a flock of geese in New York. The owner of the flock reported a large number of deaths as a result of the outbreak.

The life history of this nematode is direct. The eggs are voided in the droppings and readily hatch in the presence of moisture. The newly hatched larvae reach the infective stage within a few days; when picked up by a susceptible host, the infective larvae develop to the adult stage in the gizzard.

Young infected birds show symptoms of dullness, loss of appetite, and emaciation. The clinical symptoms are largely the result

of the improper functioning of the diseased gizzard. On post mortem examination, the inner surfaces of the gizzard, in cases of heavy infections, appear necrotic, or characterized by dead tissue; the heavy lining is loosened or sloughed in places and appears dark brown or black at the site of infection.

Large Intestinal Roundworms

One of the commonest and perhaps the most frequently seen of the parasitic roundworms of poultry is the large intestinal roundworm of chickens, *Ascaridia galli*. This parasite is very common wherever chickens are raised. It has occasionally been found in turkeys, and its occurrence in ducks and geese has been reported. Similar worms, *Ascaridia columbae* and *Ascaridia numidae*, are frequently found in considerable numbers in the intestines of pigeons and guinea fowl, respectively.

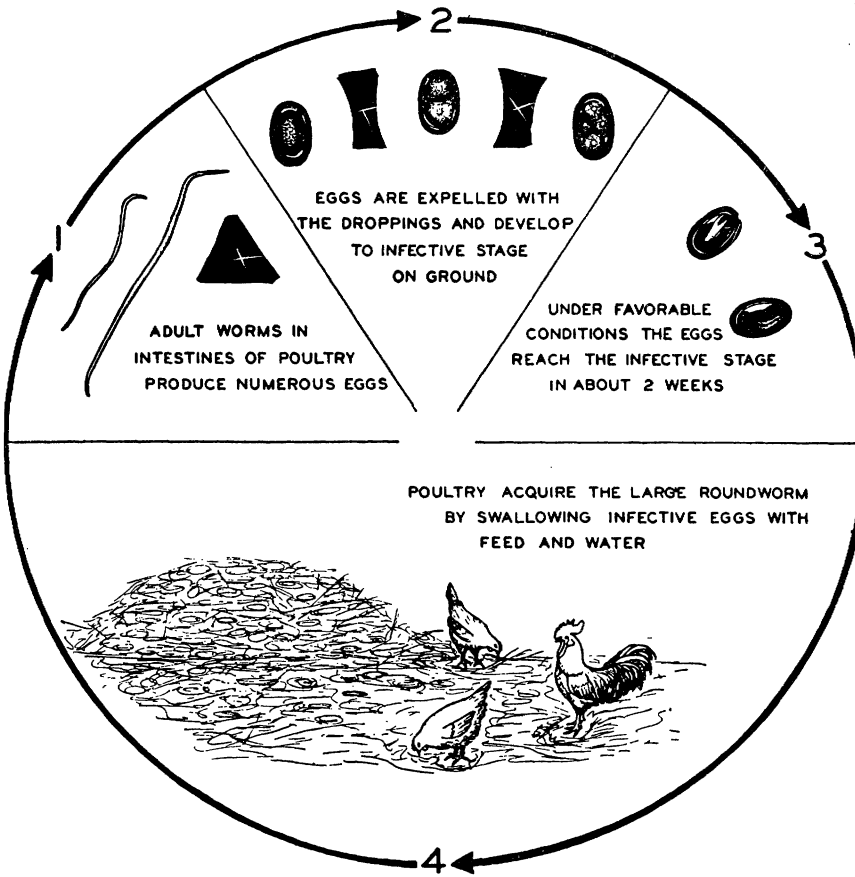

FIGURE 3.—Life-history chart of the large roundworm of the chicken, *Ascaridia galli*.

The mature worms of the three species named range in length from 1½ to 4 inches. The chicken worm is the longest and slenderest, averaging in thickness about the size of the lead of an ordinary pencil. Specimens of this ascarid have been removed on a number of occasions from broken eggs. The worms had presumably wandered up the oviduct from the intestine via the cloaca and were later incorporated in the developing egg.

The life histories of two of these ascarids have been worked out and found to be similar. Since the life history of *Ascaridia galli* (fig. 3) has been more fully discussed in the scientific literature than

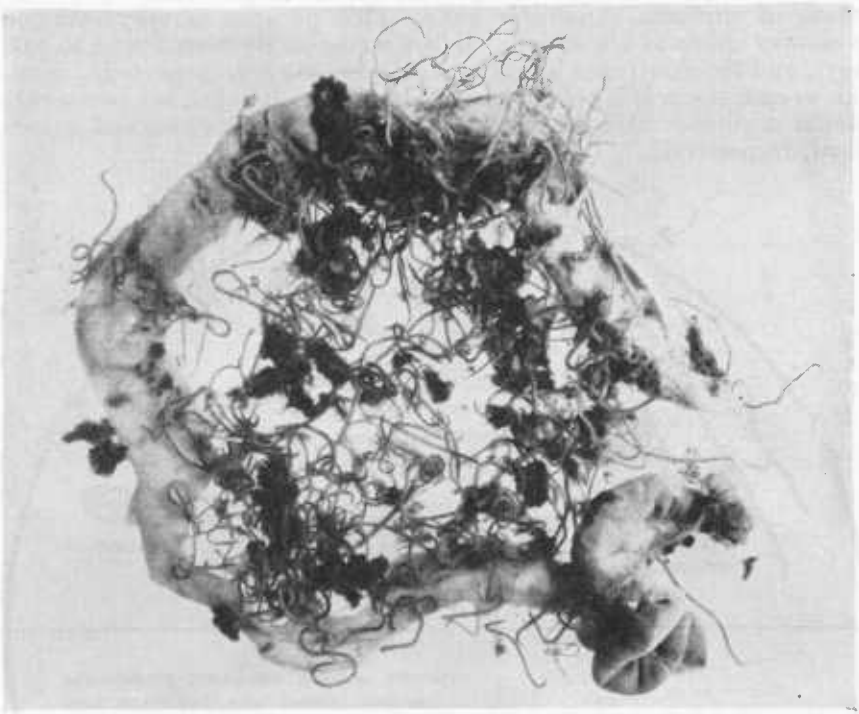

FIGURE 4.—Portion of the intestine of a bird, slit open to show the large number of roundworms.

the others, it will be only briefly reviewed here. The eggs are deposited by the female in the cavity of the intestinal tract and pass out in the droppings of the infected bird. Before these eggs become infective, they must remain outside the body of the bird host at least 2 to 3 weeks. Susceptible birds become infected by ingesting food or water containing the infective eggs. According to Itagaki (*22*), the infective eggs hatch either in the proventriculus or the duodenum. Ackert (*1*) observed that the young worms lived free in the cavity of the posterior portion of the duodenum for the first 9 days, after which they penetrated the intestinal mucous membrane and caused hemorrhages (fig. 4). By the seventeenth or eighteenth day, the

young worms have left the mucous membrane and thereafter are to be found free in the cavity of the duodenum. Maturity is reached in about 50 days.

Chickens 3 to 4 months of age or older are quite resistant to parasitism with *Ascaridia galli*. Ackert, Edgar, and Frick (*2*) stated that a relationship existed between the number of duodenal goblet cells (goblet-shaped cells on the membrane) and the mucin (the principal protoplasm in mucus) which these cells secreted and the development of the natural resistance of the growing chickens to this nematode. Since more goblet cells per area were found in the epithelial, or surface, lining of the duodenum of chickens 4 months old than in younger birds, the authors concluded that these cells were in some way responsible for the greater resistance developed by the older birds. The age at which the peak of the goblet-cell formation occurred was found to correspond very closely to the development of the maximum resistance of the chicken to the growth of the nematodes.

Animal proteins in the form of milk and meat have been shown to be important dietary supplements in the development of resistance of chickens to *Ascaridia galli*, and a diet wholly of plant origin was not found to be conducive to resistance to helminth (worm) invasion. Ackert and his coworkers have shown that foods high in vitamins A and B increase the fowl's resistance to this nematode and that the lack of the vitamin B complex definitely favors parasitism.

Birds heavily infected with *Ascaridia galli* have been found to suffer from loss of blood, reduced blood-sugar content, increased urates, shrunken thymus glands, and retarded growth, and mortality among them greatly increased. Droopiness, emaciation, and diarrhea are symptoms of a heavily parasitized condition. Death sometimes results if treatment is not given.

Small Intestinal Roundworms

Several species of hairworms, or capillarids (*Capillaria* species), occur in the small intestines and ceca of domestic fowls. Since these worms are small and hairlike in appearance and from one-half to three-fourths of an inch long, they may be easily overlooked on a casual examination of the opened intestine at autopsy. It is, therefore, necessary to resort to the microscope to make sure of their presence in case of light infections.

One species, *Capillaria columbae*, has commonly been observed in the intestines of pigeons and less commonly in the intestines of the mourning dove, chicken, and turkey. Uninfected birds become infected with this roundworm by swallowing the embryonated egg with the food and water.

Birds heavily infected with this hairworm show symptoms of emaciation, listlessness, and diarrhea. Such birds spend much of their time in a huddled position on the ground. Their feathers are ruffled and soiled around the vent, and the skin and visible mucous membranes are more or less pale. Food and water are taken sparingly. Death may occur as a result of heavy infections.

Levine (*34*) reported that the intestines of chickens heavily in-

fected with *Capillaria columbae* under experimental conditions showed a moderate thickening of the mucous membrane, which contained "reddish areas varying from pinhead hemorrhagic spots to diffuse hyperemia [excess of blood] of large portions of the mucosa." The writer has observed that the intestines of heavily infected pigeons showed extensive destruction of the mucous membrane, which was frequently completely sloughed off and contained a large quantity of fluid.

The roundworm *Ornithostrongylus quadriradiatus* may be the cause of serious intestinal disturbances in pigeons. Turtledoves and mourning doves have also been reported as hosts. The eggs of this roundworm are voided in the droppings and hatch within 19 to 24 hours. Three to four days more are required for the young larvae to become infective. When the infective larvae are swallowed by a pigeon or other susceptible host, they mature in the small intestines, and the female worms begin to deposit eggs 5 or 6 days after the larvae are ingested.

Cuvillier (*13*) reported that heavily parasitized birds become droopy and have ruffled feathers, with head and neck retracted. The birds remain squatting on the ground; if disturbed, they try to move but usually tip forward on the breast and head. Food is taken sparingly and is frequently regurgitated, along with bile-stained fluid. The birds usually drink an excessive amount of water. There is a pronounced greenish diarrhea, and the birds lose weight rapidly. Death is preceded by prostration and difficult, rapid breathing. The intestines of fatally infected birds are markedly haemorrhagic and have a green mucoid content, with masses of cast-off membrane tissue.

The cecum worm of poultry, *Heterakis gallinae*, occurs commonly in the ceca of chickens, turkeys, and possibly other domestic fowls. This worm attains a length of three-tenths to one-half inch. The life history is direct. The embryonated eggs hatch in the upper part of the intestine, and at the end of 24 hours the majority of the young worms have reached the ceca.

The chief economic importance of the cecum worm lies in its role as a carrier of the causative agent of blackhead. Graybill and Smith (*16*) discovered that blackhead can be produced by feeding large numbers of embryonated eggs of *Heterakis gallinae* removed from blackhead-infected birds. They offered the tentative hypothesis that the worms lowered the resistance of the host to the causative parasites already present in the ceca. Tyzzer and Fabyan (*47*), however, presented evidence which indicated that the protozoan parasite is incorporated in the worm egg, but they were unable to demonstrate its presence there.

Two other species of cecum worms, *Heterakis beramporia* and *H. eisolonche*, produce nodules in the ceca of chickens and pheasants, respectively.

Studies by Alicata (*5*) in Hawaii have shown that various insects, such as beetles and earwigs, serve experimentally as intermediate hosts of *Subulura brumpti*, a common pinworm of the ceca of chickens in the Hawaiian Islands.

An extremely small roundworm, *Strongyloides avium*, has been

found in the ceca and small intestine of chickens in Louisiana and Puerto Rico. The walls of the ceca and small intestine of infected birds may be greatly thickened, and a bloody diarrhea may be present. If infected chicks survive the acute stage, they may, when fully grown, show no ill effects from the parasite, even though it is present. Young chickens may die as a result of a heavy infection, but if the infection is very light little or no clinical effect has been noted.

The life history of this nematode is direct. The eggs pass out of the fowl's body in the droppings. They hatch within 18 to 24 hours, and the young worms develop in the soil into adult males and females, which shortly give rise to other young. These feed, molt, and in turn either develop into adult males and females or transform to another type of larvae known as infective larvae. When these infective larvae are swallowed by a susceptible host, infection results. Unlike most species of nematodes, the parasitic cycle of *Strongyloides avium* consists of females only.

The Eye Worm

The eye worm of poultry, *Oxyspirura mansoni* is found only in Florida and Louisiana. The white worms are found beneath the nictitating membrane, or third eyelid, sometimes in large numbers.

Studies by Sanders (*41*) in Florida showed that the cockroach *Leucophala surinamensis* is the intermediate host of the chicken eye worm. The eggs of the female worms are washed down the tear ducts, swallowed, and pass to the exterior in the droppings. The cockroach ingests the eggs or newly hatched larvae, and the latter develop to the infective stage in the body cavity of the insect. When the cockroach is subsequently eaten by a chicken or other susceptible host, the infective larva is freed in the crop and passes up the esophagus to the mouth, and then through the nasolachrymal duct (the tear duct) to the eye. Affected birds show signs of uneasiness and scratch at the eyes, which exhibit an acute inflammation accompanied by an abundant secretion of tears. The nictitating membrane is swollen, projects slightly beyond the eyelids at the corners of the eye, and is kept in continual motion as if to remove some foreign body from the eye. The eyelids sometimes become stuck together, and a white cheesy matter collects beneath them. Occasionally severe ophthalmia (inflammation of the eye) develops, and the eyeball may be destroyed if treatment of some sort is not resorted to. When this stage is reached the worms are no longer to be found in the eye. Severely affected birds eat very little, decline in strength, become anemic, and may die within a few weeks.

Gapeworms

Gapeworm infection, commonly known as gapes, is caused by roundworms that live in the windpipe. These worms, *Syngamus trachea*, are sometimes called red worms because of their red color or forked worms because the males and females so firmly adhere to one another that they appear like the letter Y. The male worm attains

a length of about one-fifth of an inch, while the female reaches a length of nearly 1 inch.

This parasite has a direct life history. Eggs are coughed up from the windpipe and swallowed by the bird. They pass out in the droppings and develop, and some of them hatch. Fowls become infected by swallowing either the infective eggs or the young roundworms that hatch from them. Earthworms may swallow the infective eggs or the young roundworms and are then a source of infection to fowls that swallow them.

The worms clog the windpipe of young poultry, causing them to sneeze, cough, and gape for air (fig. 5). An extensive irritation of the mucous lining of the windpipe results from the bloodsucking activities of the gapeworms, and coughing is apparently caused by

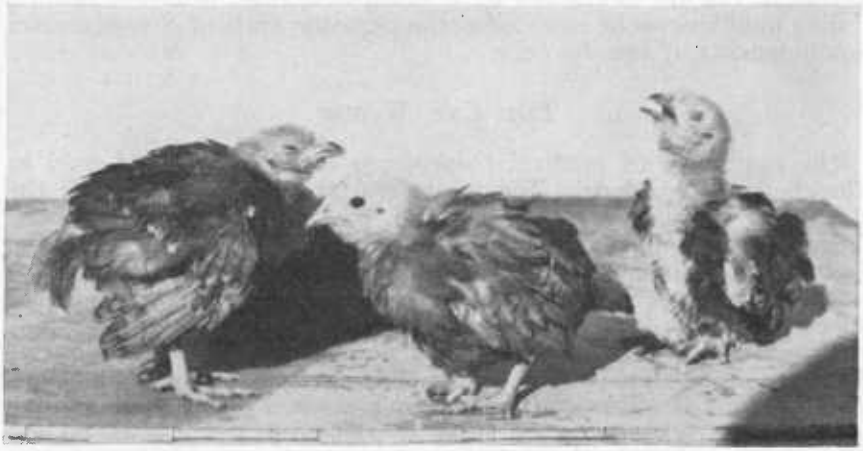

FIGURE 5.—Young chickens infected with gapeworms. The chicken on the right is gaping as a result of obstruction of the trachea by the worms.

this irritation. Similar symptoms result from bronchitis and laryngotracheitis, but if gapeworms are responsible they can be readily found by destroying a sick bird and slitting open the windpipe. The red, Y-shaped worms, if present, are usually found in the lower half of the windpipe.

Lesions or nodules are usually found at the point of attachment of the male worms only. Hence, it is believed that the male worm usually remains permanently attached to the tracheal wall, while the female worm loosens her hold from time to time and selects a new feeding place. Nodule formation occurs frequently in the tracheas of infected turkeys but is rarely seen in infected chicks because the latter rarely remain infected long enough for it to take place.

Affected birds become weak and emaciated and spend much of their time huddled on the floor with the eyes closed and the head drawn back against the body. The head is regularly thrown forward and upward and the mouth is opened wide to draw in air, or the head may be given a convulsive shake in an attempt to loosen the obstruc-

tion in the windpipe so that normal breathing may be resumed. Sudden death is due primarily to suffocation caused by the mechanical obstruction of the windpipe by the rapidly growing worms and the accumulation of secreted mucus.

Wehr (*52*) observed that young turkey poults usually develop gapeworm symptoms in approximately 7 or 8 days, whereas young chickens do not ordinarily show symptoms of gaping and coughing until 10 to 14 days after experimental infection. The turkey poults likewise begin to die from gapeworm infections sooner than young chickens. Characteristic gapeworm symptoms of coughing and gaping have not been observed in guinea fowl and ducks. Young pheasants, however, suffer from the disease to an extent comparable to that of young chicks and turkey poults. Older birds, unless heavily infected, usually show only mild symptoms or none at all. Chickens more than 10 weeks of age seldom harbor gapeworms under natural conditions. It has been reported, however, that in Ceylon gapeworms occur commonly in fowls of all ages, even up to 3 years. The role played by wild birds in the spread of gapeworm disease is still questionable.

LITERATURE CITED

(1) ACKERT, JAMES E.
1931. THE MORPHOLOGY AND LIFE HISTORY OF THE FOWL NEMATODE ASCARIDIA LINEATA (SCHNEIDER). Parasitology 23: 360–379, illus.

(2) ——— EDGAR, S. A., and FRICK, L. P.
1939. GOBLET CELLS AND AGE RESISTANCE OF ANIMALS TO PARASITISM. Amer. Micros. Soc. Trans. 58: 81–89.

(3) ——— and GRAHAM, G. L.
1935. THE EFFICACY OF CARBON TETRACHLORIDE IN ROUNDWORM CONTROL. Poultry Sci. 14: 228–231, illus.

(4) ——— and REID, W. M.
1937. AGE RESISTANCE OF CHICKENS TO THE CESTODE RAILLIETINA CESTICILLUS (MOLIN). (Abstract) Jour. Parasitol, 23: 558.

(5) ALICATA, JOSEPH E.
1939. PRELIMINARY NOTE ON THE LIFE HISTORY OF SUBULURA BRUMPTI, A COMMON CECAL NEMATODE OF POULTRY IN HAWAII. (Research note) Jour. Parasitol. 25: 179–180, illus.

(6) ——— and JONES, MYRNA F.
1933. THE DUNG BEETLE, ATAENIUS COGNATUS, AS THE INTERMEDIATE HOST OF HYMENOLEPIS CANTANIANA. (Abstract of paper) Jour. Parasitol. 19: 244, illus.

(7) ALLEN, ENA A.
1940. A REDESCRIPTION OF TRICHOMONAS GALLINARUM MARTIN AND ROBERTSON, 1911, FROM THE CHICKEN AND TURKEY. Helminthol. Soc. Wash. Proc. 7: 65–68, illus.

(8) ———
1941. MACROSCOPIC DIFFERENTIATION OF LESIONS OF HISTOMONIASIS AND TRICHOMONIASIS IN TURKEYS. Amer. Jour. Vet. Res. 2: 214–217, illus.

(9) [BARBER, L. B.]
1916. LIVE STOCK DISEASE INVESTIGATIONS. Guam Agr. Expt. Sta. Rpt. 1915: 25–41, illus.

(10) BISHOPP, F. C.
1927. THE FOWL TICK AND HOW PREMISES MAY BE FREED FROM IT. U. S. Dept. Agr. Farmers' Bul. 1070, 14 pp., illus. (Revised.)

(11) CRAM, ELOISE B.
1926. A PARASITIC NEMATODE AS THE CAUSE OF LOSSES AMONG DEMESTIC GEESE. North Amer. Vet. 7: 27–29, illus.
(12) ———
1936. SPECIES OF CAPILLARIA PARASITIC IN THE UPPER DIGESTIVE TRACT OF BIRDS. U. S. Dept. Agr. Tech. Bull. 516, 28 pp., illus.
(13) CUVILLIER, EUGENIA.
1937. THE NEMATODE, ORNITHOSTRONGYLUS QUADRIRADIATUS, A PARASITE OF THE DOMESTICATED PIGEON. U. S. Dept Agr. Tech. Bull. 569, 36 pp., illus.
(14) GIERKE, A. G.
1933. TRICHOMONIASIS OF THE UPPER DIGESTIVE TRACT OF CHICKENS. Calif. Dept. Agr., Monthly Bul. 22: 205–208, illus.
(15) GRASSI, [B.], and ROVELLI, [G.]
1888. DÉVELOPPEMENT EXPERIMENTAL DU TÆNIA PROGLOTTINA DAV. (Revue) Rec. de Méd. Vét. 65: 675–676.
(16) GRAYBILL, H. W., and SMITH, THEOBALD.
1920. PRODUCTION OF FATAL BLACKHEAD IN TURKEYS BY FEEDING EMBRYONATED EGGS OF HETERAKIS PAPILLOSA. Jour. Expt. Med. 31: 647–655.
(17) HALL, MAURICE C., and SHILLINGER, JACOB E.
1923. MISCELLANEOUS TESTS OF CARBON TETRACHLORIDE AS AN ANTHELMINTIC. Jour. Agr. Res. 23: 163–192.
(18) HARWOOD, PAUL D., and LUTTERMOSER, GEORGE W.
1938. THE INFLUENCE OF INFECTIONS WITH THE TAPEWORM, RAILLIETINA CESTICILLUS, ON THE GROWTH OF CHICKENS. Helminthol. Soc. Wash. Proc. 5: 60–62.
(19) HAWN, M. C.
1937. TRICHOMONIASIS OF TURKEYS. Jour. Infect. Dis. 61: [184]–197.
(20) HINSHAW, W. R., and MCNEIL, E.
1941. CARRIERS OF HEXAMITA MELEAGRIDIS. Amer. Jour. Vet. Res. 2: 453–458.
(21) ——— MCNEIL, E., and KOFOID, C. A.
1938. THE RELATIONSHIP OF HEXAMITA SP. TO AN ENTERITIS OF TURKEY POULTS. Cornell Vet. 28: 281–293.
(22) ITAGAKI, SHIRO.
1927. ON THE LIFE HISTORY OF THE CHICKEN NEMATODE, ASCARIDIA PERSPICILLUM. 3d World's Poultry Cong. Proc., pp. 339–344, illus.
(23) JOHNSON, E. P.
1939. A METHOD OF RAISING TURKEYS IN CONFINEMENT TO PREVENT PARASITIC DISEASES. Va. Agr. Expt. Sta. Bul. 323, 16 pp.
(24) ——— UNDERHILL, G. W., COX, J. A., and THRELKELD, W. L.
1938. A BLOOD PROTOZOON OF TURKEYS TRANSMITTED BY SIMUDIUM NIGROPARVUM (TWINN). Amer. Jour. Hyg. 27: 649–665, illus.
(25) JONES, MYRNA F.
1936. METROLIASTHES LUCIDA, A CESTODE OF GALLIFORM BIRDS, IN ARTHROPOD AND AVIAN HOSTS. Helminthol. Soc. Wash. Proc. 3: 26–30, illus.
(26) ——— and HORSFALL, M. W.
1935. ANTS AS INTERMEDIATE HOSTS FOR TWO SPECIES OF RAILLIETINA PARASITIC IN CHICKENS. Jour. Parasitol. 27: 442–443.
(27) ——— and HORSFALL, MARGERY W.
1936. THE LIFE HISTORY OF A POULTRY CESTODE. Science 83: 303–304.
(28) JOYEUX, CHARLES, and BAER, JEAN GEORGES.
1937. RECHERCHES SUR L'EVOLUTION DES CESTODES DE GALLINACÉS. [Paris] Acad. des Sci. Compt. Rend. 205: 751–753.
(29) JUNGHERR, ERWIN.
1927. TWO INTERESTING TURKEY DISEASES. Amer. Vet. Med. Assoc. Jour. 71: 636–640, illus.
(30) KOTLAN, A., and CHANDLER, W. L.
1925. A NEWLY RECOGNIZED FLUKE DISEASE (PROSTHOGONIMIASIS) OF FOWLS IN THE UNITED STATES. Amer. Vet. Med. Assoc. Jour. 67: 756–763, illus.
(31) ——— and CHANDLER, W. L.
1927. ON THE ROLE PLAYED BY DRAGONFLIES IN THE TRANSFER OF PROSTHOGONIMUS. Amer. Vet. Med. Assoc. Jour. 70: 520–524.

(32) LEVINE, NORMAN D., BOLEY, L. E., and HESTER, H. R.
1941. EXPERIMENTAL TRANSMISSION OF TRICHOMONAS GALLINAE FROM THE CHICKEN TO OTHER BIRDS. Amer. Jour. Hyg., Sect. C, 33: 23–32.
(33) ——— and BRANDLY, C. A.
1939. A PATHOGENIC TRICHOMONAS FROM THE UPPER DIGESTIVE TRACT OF CHICKENS. Amer. Vet. Med. Assoc. Jour. 95: 77–78, illus.
(34) LEVINE, P. P.
1938. INFECTION OF THE CHICKEN WITH CAPILLARIA COLUMBAE (RUD.). Jour. Parasitol. 24: [45]–52.
(35) MACY, RALPH W.
1934. PROSTHOGONIMUS MACRORCHIS N. SP., THE COMMON OVIDUCT FLUKE OF DOMESTIC FOWLS IN THE NORTHERN UNITED STATES. Amer. Micros. Soc. Trans. 53: 30–34, illus.
(36) ———
1934. STUDIES ON THE TAXONOMY, MORPHOLOGY, AND BIOLOGY OF PROSTHOGONIMUS MACRORCHIS MACY, A COMMON OVIDUCT FLUKE OF DOMESTIC FOWLS IN NORTH AMERICA. Minn. Agr. Expt. Sta. Tech. Bul. 98. 71 pp., illus.
(37) MCCULLOCH, ERNEST C., and NICHOLSON, LYLE G.
1940. PHENOTHIAZINE FOR THE REMOVAL OF HETERAKIS GALLINAE FROM CHICKENS. Vet. Med. 35: 398–400, illus.
(38) OLSEN, MARLOW W., and ALLEN, ENA A.
1940. TREATMENT OF CECAL AND LIVER TRICHOMONIASIS IN TURKEYS BY FEVER THERAPY. (Preliminary paper) Soc. Expt. Biol. and Med. Proc. 45: 875–876.
(39) O'ROKE, EARL C.
1934. A MALARIA-LIKE DISEASE OF DUCKS CAUSED BY LEUCOCYTOZOON ANATIS WICKWARE. Mich. Univ. School Forestry and Conserv. Bul. 4, 44 pp., illus.
(40) ROBERTS, F. H. S.
1940. A PRELIMINARY NOTE ON THE EFFICIENCY OF PHENOTHIAZINE AGAINST SOME POULTRY HELMINTHS. Austral. Vet. Jour. 16: 172–174.
(41) SANDERS, D. A.
1928. MANSON'S EYEWORM OF POULTRY. Amer. Vet. Med. Assoc. Jour. 72: 568–584, illus.
(42) SKIDMORE, LOUIS V.
1932. LEUCOCYTOZOON SMITHI INFECTION IN TURKEYS AND ITS TRANSMISSION BY SIMULIUM OCCIDENTALE TOWNSEND. Zentbl. f. Bakt. [etc.] Originale (I) 125: 329–335, illus.
(43) STABLER, ROBERT M.
1938. THE SIMILARITY BETWEEN THE FLAGELLATE OF TURKEY TRICHOMONIASIS AND T. COLUMBAE IN THE PIGEON. Amer. Vet. Med. Assoc. Jour. 93: 33–34, illus.
(44) ———
1938. TRICHOMONAS GALLINAE RIVOLTA (1878) THE CORRECT NAME FOR THE FLAGELLATE IN THE MOUTH, CROP AND LIVER OF THE PIGEON. (Research note) Jour. Parasitol. 24: 553–554.
(45) TYZZER, ERNEST EDWARD.
1927. ENTERO-HEPATITIS IN TURKEYS AND ITS TRANSMISSION THROUGH THE AGENCY OF HETERAKIS VESICULARIS. 3d World's Poultry Cong. Proc., pp. 286–290, illus.
(46) ———
1934. STUDIES ON HISTOMONIASIS, OR "BLACKHEAD" INFECTION, IN THE CHICKEN AND TURKEY. Amer. Acad. Arts and Sci. Proc. 69: [189]–264, illus.
(47) ——— and FABYAN, M.
1920. FURTHER STUDIES ON "BLACKHEAD" IN TURKEYS, WITH SPECIAL REFERENCE TO TRANSMISSION BY INOCULATION. Jour. Infect. Dis. 27: [207]–239, illus.
(48) UNDERHILL, G. W.
1939. TWO SIMULIIDS FOUND FEEDING ON TURKEYS IN VIRGINIA. Jour. Econ. Ent. 32.: 765–768.

(49) VOLKMAR, FRITZ.
1930. TRICHOMONAS DIVERSA N. SP. AND ITS ASSOCIATION WITH A DISEASE OF TURKEYS. Jour. Parasitol. 17: [85]–89, illus.
(50) WEHR, EVERETT E.
1936. EARTHWORMS AS TRANSMITTERS OF CAPILLARIA ANNULATA, THE "CROP-WORM" OF CHICKENS. North Amer. Vet. (8): 18–20, illus.
(51) ———
1937. RELATIVE ABUNDANCE OF CROP WORMS IN TURKEYS: MACROSCOPIC DIFFERENTIATION OF SPECIES. Vet. Med. 32: 230–233, illus.
(52) ———
1939. THE GAPEWORM AS A MENACE TO POULTRY PRODUCTION. 7th World's Poultry Cong. Proc., pp. 267–270.
(52a) ———
1939. STUDIES ON THE DEVELOPMENT OF THE PIGEON CAPILLARID, CAPILLARIA COLUMBAE. U. S. Dept. Agr. Tech. Bul. 679, 19 pp., illus.
(53) ——— HARWOOD, PAUL D., and SCHAFFER, JACOB M.
1939. BARIUM ANTIMONYL TARTRATE AS A REMEDY FOR THE REMOVAL OF GAPEWORMS FROM CHICKENS. Poultry Sci. 18: 63–65.

At the time this book went to press, the drugs and other materials mentioned in various articles—chiefly as disinfectants, insecticides, and anthelmintics—were still available for veterinary and medical use. Under war conditions, however, it is possible that some of these materials may become scarce or unavailable. In that case, the reader should obtain professional advice from the Department of Agriculture, the State experiment station, a local veterinarian, or the county agent as to available substitutes.